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## Little's disease

Every student is taught about Little's spastic diplegia, but how many know that William John Little (1810–94) was himself afflicted with a congenital equinus deformity of his left foot? He was founder of the Royal Orthopaedic Hospital and Senior Physician to the London Hospital. His several disquisitions<sup>1-3</sup> are quite brilliant and repay study in full.

"I have witnessed so many cases of deformity, mental and physical, traceable to causes operative at birth, that I consider the subject worthy of notice<sup>3</sup> of the Obstetrical Society." He mentions 200 cases encountered in 20 years of orthopaedic practice. He stressed the "larger proportion of dead, stillborn, apoplectic, or asphyxiated at birth have been rendered so by interruption of the proper placental relation of the foetus to the mother, and non-substitution of pulmonary respiration, than from direct mechanical injury to the brain and spinal cord." The consequences were "internal congestions, capillary extravasations, serous effusions which correspond with. . . asphyxia, suspended animation, apoplexy, torpidity, tetanic spasms, convulsions of newborn children, and the spastic rigidity, paralysis, and idiocy subsequently witnessed."

"The flexors and abductors of the thighs, flexors of the knees, and the gastrocnemii preponderate... thighs cannot be completely abducted or extended, the knees cannot be straightened, nor the heels applied to the ground. The elbows are semi-flexed, wrists partially flexed and pronated and fingers incapable of perfect voluntary direction... upper extremities sometimes appear unaffected... Muscles of speech are commonly involved,... articulation is slow and difficult... in the majority of cases the intellect suffers—from the slightest impairment up to entire imbecility."

Little relates presentations with convulsions, opisthotonus or laryngismus in the early days and the delay in recognising paralysis until the child starts to walk in milder cases; he describes the deformities of posture, trunk and the impediment in walking. He had sparse necropsy evidence "Case LX showing cicatrised apoplexies on surface and interior surfaces of the brain... effusion of blood in both ventricles of brain—a true apoplexy (case XLI—a precipitate labour). But, curiously, he concluded "that the spinal meningitic and myelitic affections may play a considerable part in the phenomena of spastic rigidity." His finale is a brief but optimistic reference to "therapeutic effects producing amelioration surprising to those who have not watched such cases."

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